The Mouth

Introduction

The lessons in this first content island, Digestion and Absorption: Start to Finish, explore the different segments of the GI tract. This is a large island. It is about how the complex interactions between the gut tube and its accessory organs facilitate digestion, absorption, and elimination, with a huge emphasis on the first two. The GI tract is made from endoderm, from the gut tube in embryologic development. It is primarily designed for the digestion and absorption of ingested contents. As we will see, the gut tube features a single histologic arrangement—four layers—for the majority of its length. But not in the mouth.

The Mouth is the first lesson of the Digestion and Absorption: Start to Finish island and the first lesson in Gastroenterology only because it is the first segment of the GI tract used in the process of eating. We will reveal more about the embryogenesis, histology, and function of the GI tract as we progress through the GI module, each lesson building on those that came before it. And because the mouth is so unlike the rest of the GI tract, and the rest of the GI tract regions are so similar to each other, it is convenient to get this one out of the way first. You will not learn much in this lesson that is useful for the rest of the island or the module, but it is stuff you have to know. And it is where digestion begins, both mechanical and chemical.

We will discuss the tongue, palate, salivation, and the initiation of swallowing. Like the GI tract itself, the lessons are meant to be experienced in a specific order. This mouth lesson ends with the initiation of swallowing, which is where the esophagus lesson picks up.

Vocabulary and Anatomy of the Mouth

This first section is meant only to identify and name the structures in the mouth—limited details and no functionality. What we call the mouth is better named the oral cavity. It is the space where food is chewed and then passed backward to the esophagus. Most of the oral cavity is occupied by the tongue (intrinsic muscles of the tongue) and the muscles that move the tongue (extrinsic muscles of the tongue). There are also salivary glands, teeth, and tonsils in the oral cavity. In the back of the oral cavity is the uvula, which is connected to the palatine arches. Behind the palatine arches and the tongue is a space where food is projected, called the isthmus.

At the top of the oral cavity is the palate, which is divided into a hard palate (bone) and soft palate (not bone). Above the palate relative to the oral cavity lies the nasal cavity. The oral cavity opens into the pharynx, and where the oral cavity meets the pharynx, it is called the oropharynx. The topmost region of the pharynx is located at the rear of the nasal cavity, above the palate, and is called the nasopharynx. Below the palate, but above the opening to the larynx, is the oropharynx. Below the larynx is the laryngopharynx.

The palatine arches comprise the palatoglossus muscle covered by pharyngeal mucosa. The oral cavity (where there aren't teeth, bones, or tongue muscle), nasopharynx, oropharynx, laryngopharynx, and the opening of the esophagus are all lined with pharyngeal epithelium. The pharyngeal epithelium is a nonkeratinized stratified squamous epithelium. Many people use "mucosa" and "epithelium" interchangeably (we'll discuss why they should not be interchangeable in the next lesson). When talking about the shiny glistening lining of the inside of your mouth and throat, say mucosa. When talking about the histologic appearance and function, and, therefore, the risk of infection and cancer, say epithelium.

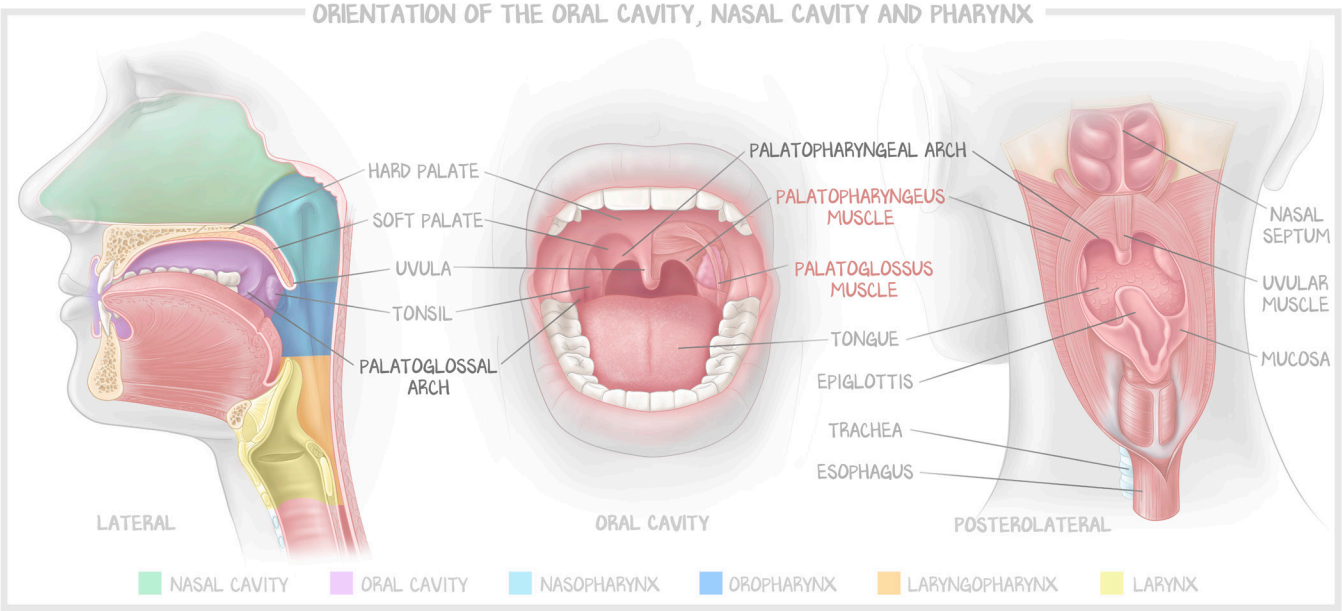

Figure 1.1: Orientation of the Oral Cavity, Nasal Cavity, and Pharynx
This illustration gives you multiple perspectives of the nasal and oral cavities and their related pharynxes as well as a glimpse into the anatomy of the trachea and esophagus. This is meant to be a reference tool. As you progress, come back to this image to help orient yourself.

We dive into pharyngeal pathology in the Pulmonary module. We just wanted to start you off in GI with all the right parts. Notice that nothing was bolded. Everything in this section is necessary in order to comprehend what follows in this lesson, but none of this warrants an evaluation question or a flashcard. Refer back to these paragraphs and these illustrations as you progress through this lesson.

Anatomy and Embryogenesis of the Palate

The normal, intact roof of the mouth (the one palate) can be divided into two continuous palates—the hard palate and the soft palate. The **hard palate** is bone covered with mucosa. It makes up the roof of the mouth and the floor of the nasal cavity. The **soft palate** is mucosa only. The uvula is the end of the soft palate. The hard palate has the palatine rugae—you can feel these ridges with your tongue. More importantly, the hard palate **extends as far back as there are teeth**. The teeth, being bone, come from the hard palate, which is also bone.

In embryogenesis, there are two palates. These two palates DO NOT have any overlap with the two final, adult palates. The **primary palate** forms from the medial nasal prominence to become part of the hard palate (bone covered by mucosa) and the front four teeth (bone only). The **secondary palate** forms from the maxillary prominences to become the remainder of the hard palate (bone covered by mucosa) as well as the entirety of the soft palate (mucosa only). These are embryologic structures and, again, DO NOT have any overlap with the hard and soft palate. Keep the adult intact anatomic structures (hard and soft) separate from the developing embryonic structures (primary and secondary) in your mind.

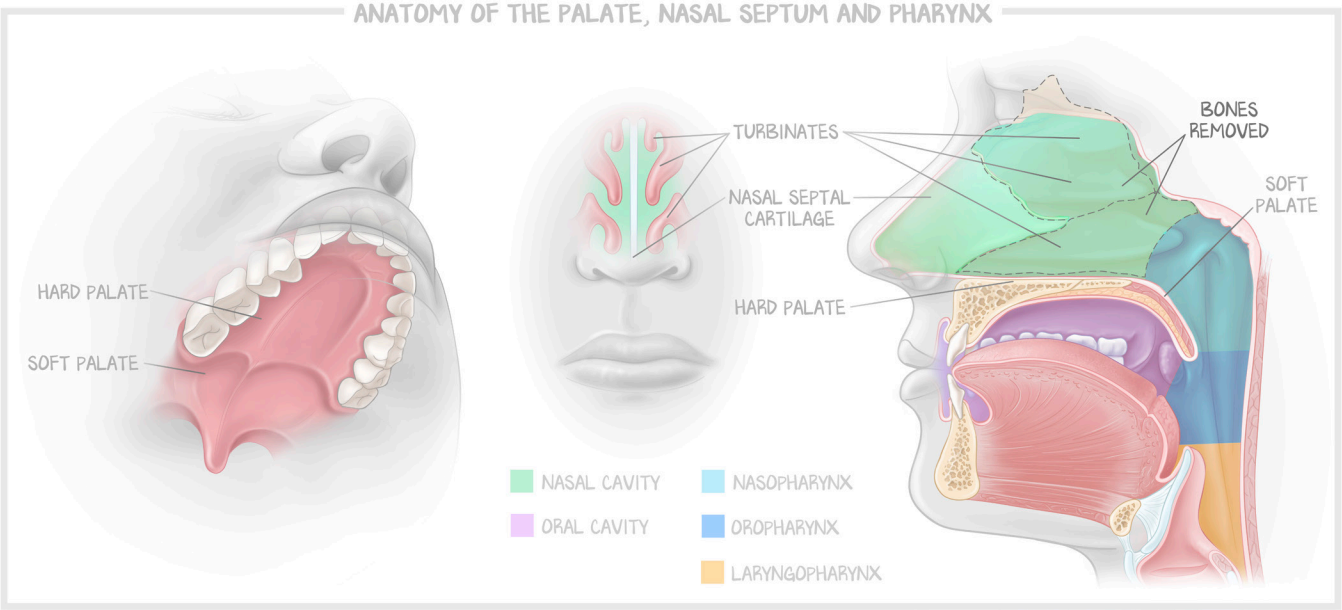

Figure 1.2: Anatomy of the Palate, Nasal Septum, and Pharynx
The relative anatomy of the hard and soft palates and the nasal septum, as well as the nasopharynx and oropharynx.

Embryology is hard. There are things. These things could be called ridges, folds, glops of cells, placodes, arches, or anything else. Their name changes as embryogenesis progresses. For this discussion, everything that matters is named a prominence. There are ten prominences, five on each side. They are masses of cells that replicate to form the outside of the face, the palate, and the nasal cavity, and any other structures we will show you don't matter for this discussion. The prominences have to grow in size and fuse together. We are going to start with the full list, but then pare it down because the congenital diseases that actually happen in live births involve very few fusion failures. There are five prominences: mandibular, maxillary, frontonasal, lateral nasal, and medial nasal. Refer to Figure 1.3 as you read.

The **mandibular prominences** must grow and fuse to form the bottom half of the jaw. The fused mandibular prominences must also fuse with the ipsilateral maxillary prominence to make the cheeks. Failure to do so is not seen in a live birth and represents a catastrophic failure very early in development. We will not consider the mandibular prominences again.

The **frontonasal prominences** are the origin of the medial and lateral nasal prominences. We will not consider the frontonasal prominences again.

The **lateral nasal prominences** just get to stay lateral and be nasal. They are the lateral sides of our nose, the alar cartilages. They do not fuse, and so no disease can result from their failure to fuse. We will not consider the lateral nasal prominences again.

The **medial nasal prominences** need to fuse with each other. They are going to form the bridge of the nose, philtrum of the lip, primary palate, and nasal septum. All of those things come from and are synonymous with the medial nasal prominences. There are disease states that arise from a failure of the medial nasal prominences to fuse, so we will continue to consider them.

Then there are these other guys, the **maxillary prominences**. They cause all the fuss. If the maxillary prominences don't fuse to anything, they will still become the maxillary bones. They should fuse with the mandibular prominences to make our cheeks (and they usually do). But the maxillary prominences must also fuse with the medial nasal prominences and each other to make the upper lips and the majority of the palate. Their NOT fusing is what causes cleft lip and cleft palate. The palatine shelves and maxillary prominences are the same thing.

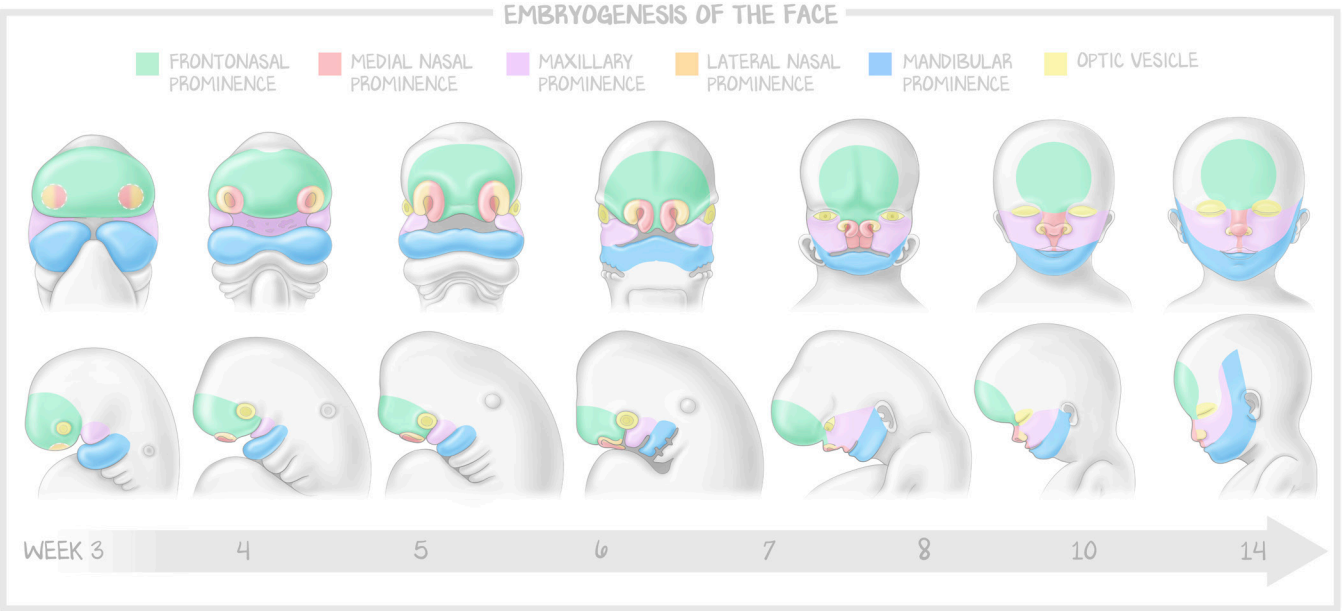

Figure 1.3: Embryogenesis of the Face
This color-coding of the prominences is continued throughout subsequent illustrations and enables the reader to follow along with the text. The color-coding will not change, although the names of the structures change as they develop.

Now we turn our attention to the successful formation of the palate and septum. The medial nasal prominences have already fused. The medial nasal prominences fuse to their ipsilateral maxillary prominences in the front, whereas the maxillary prominences fuse with one another along the length of the oral cavity. The maxillary prominences form across the oral cavity as palatine shelves. (See Figure 1.4.)

In **primary palate fusion**, the maxillary prominences (as palatine shelves) fuse with the medial nasal prominences (as the primary palate). This will create the upper lip and connect it to the medial nasal prominence (as the philtrum). This will also connect the bone of the hard palate made by the maxillary prominences to the bone of the hard palate made by the medial nasal prominences.

In **secondary palate fusion**, the maxillary prominences (as palatine shelves) must fuse with each other. They start that fusion immediately after primary palate fusion (in time and space), then zip up the palate from the front towards the back. The intact palate is complete when that zipping up reaches the uvula.

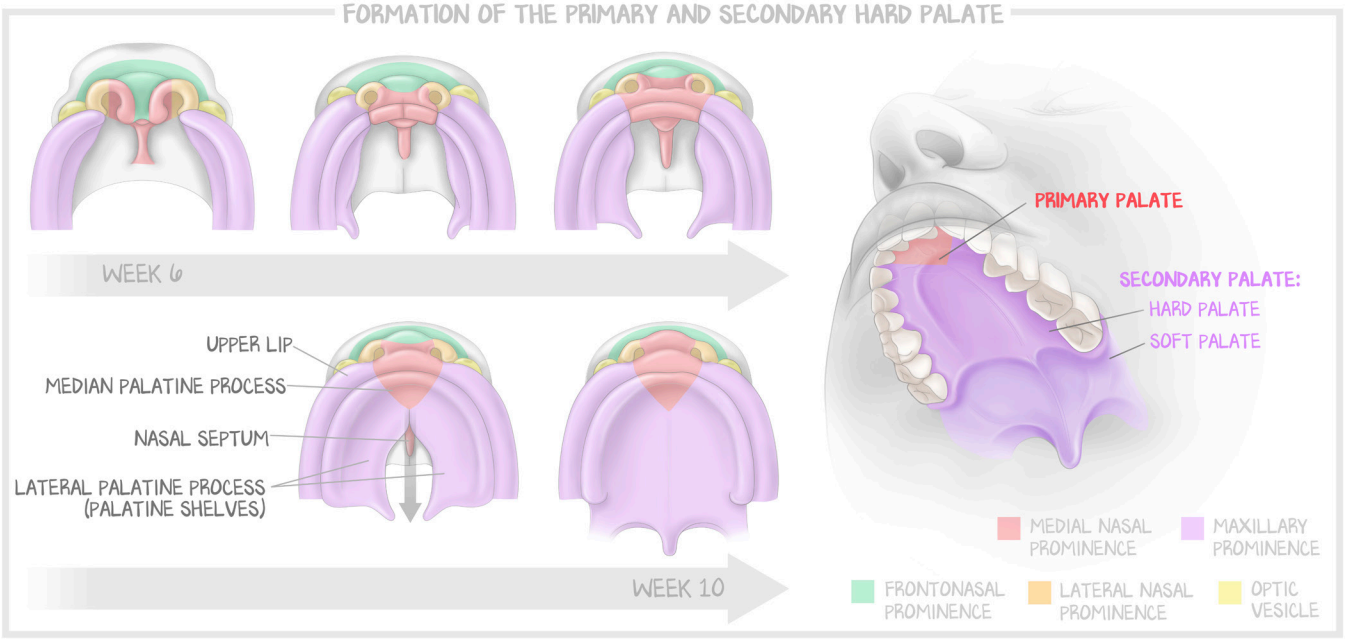

Figure 1.4: Formation of the Primary and Secondary Hard Palate

At the same time, the medial nasal prominences grow back into the nasal cavity above the palatine shelves as the nasal septum. **The fusion of the nasal septum** with the palate is the fusion of the medial nasal prominences with the maxillary prominences. As the maxillary prominences (as palatine shelves) zip up the palate front to back, they also fuse with the nasal septum (as medial nasal prominences). This is less important to understand, but we want you to really feel the concept of "everything is just purple and orange fusing." (See Figure 1.5.)

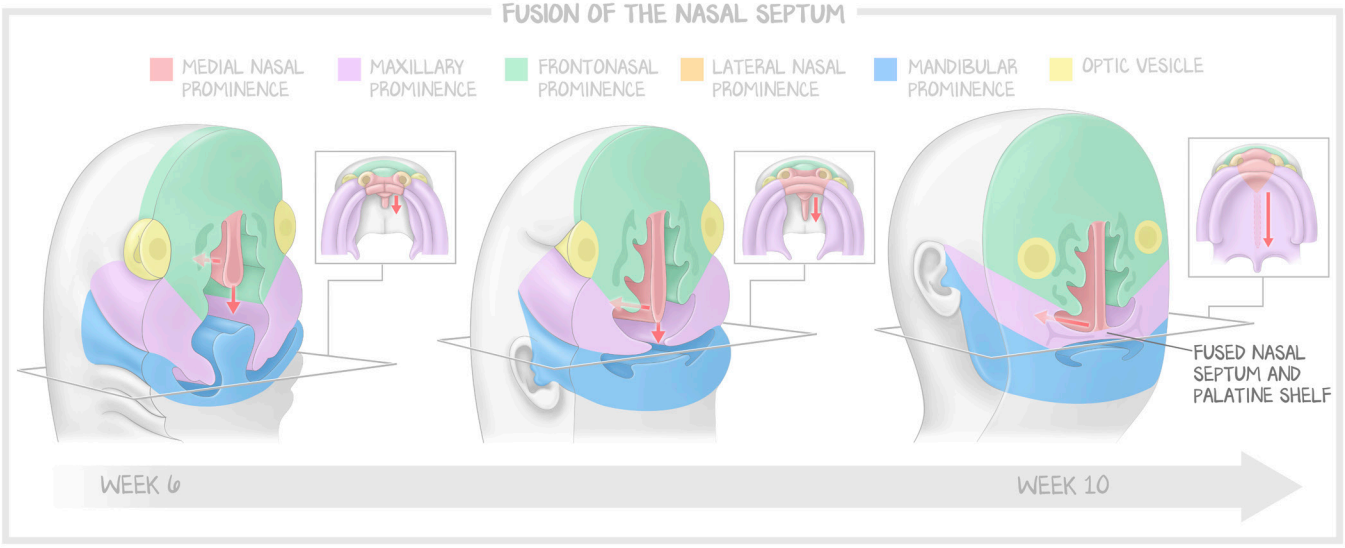

Figure 1.5: Fusion of the Nasal Septum

We turn now to defects in fusion—cleft lip and cleft palate. Use Figure 1.6 as you move through the text.

A **cleft lip** is a discontinuous upper lip. The medial nasal prominences will make the philtrum. The maxillary prominences will make the rest of the upper lip. Because the medial nasal prominences never fail to fuse with each other, there's only one way cleft lip can happen: **the failure of a maxillary prominence to fuse with its medial nasal prominence**. A cleft lip can occur in conjunction with a cleft palate or in isolation.

A **cleft palate** can result from either primary or secondary palate fusion failure. In **primary failure** (fusion of one maxillary prominence with its medial nasal prominence, just as in cleft lip), there is both a cleft lip and a cleft palate, the cleft being contiguous between the skin and bone. In **secondary palate failure** (fusion of the maxillary prominences with each other), there is no lip defect. Instead, the defect will be seen midline and posterior to the contribution of the medial nasal prominence (as the primary palate). Because the maxillary prominences (as palatine shelves) zip up front to back, the cleft will be from the point of defect back to the uvula. The most minor form of this defect is a bifid uvula (failure to fuse at the most posterior of the palate). The worst form of secondary palate failure is the absence of the fusion of the medial nasal prominences (as the primary palate), causing a cleft palate of the entire length of the oral cavity.

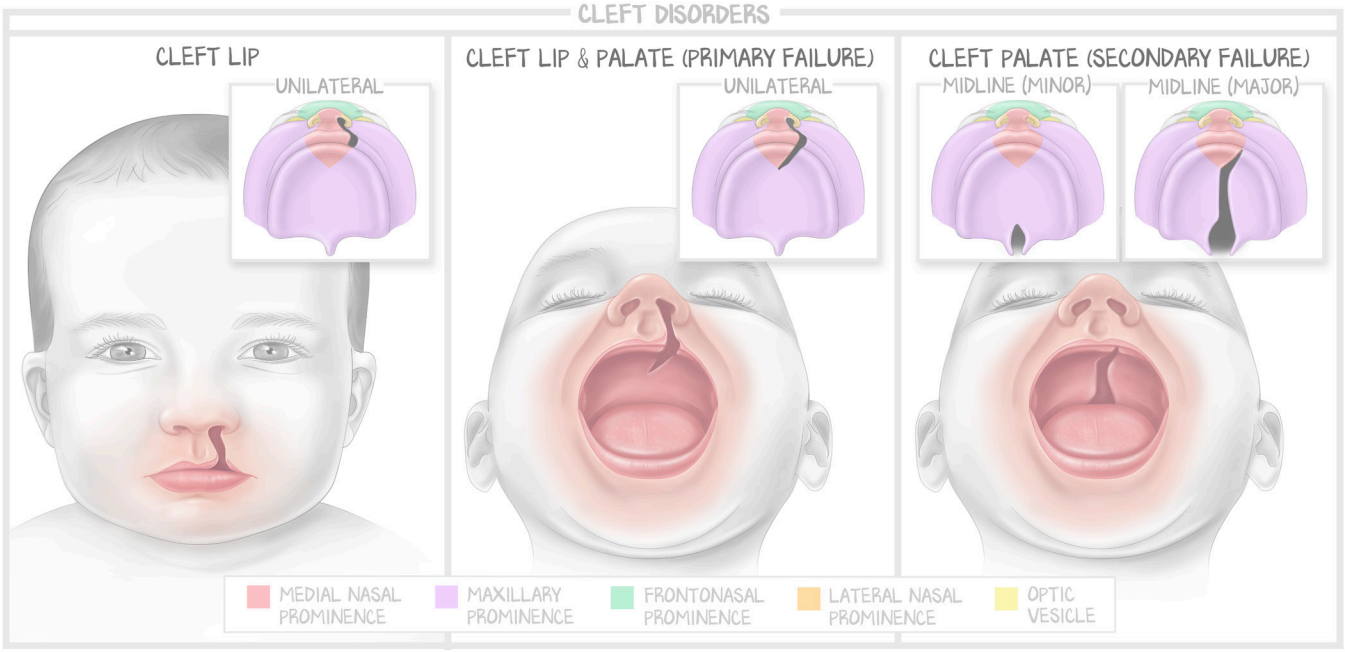

Figure 1.6: Cleft Disorders
Always the result of a failure of the maxillary prominences to fuse, there can be a unilateral cleft lip, unilateral cleft lip and palate, or midline cleft palate. Of course, the failure to fuse can occur on both sides, although that presentation is very rare.

An infant feeds by drawing milk from a nipple (either a bottle or mom's breast), then swallowing that milk. Baby instinctually coordinates breathing with eating. When baby swallows, no breathing. When drawing milk into its mouth, nose breathing. To draw milk out, baby must be able to **latch**—create a seal around the nipple—and **suck**—generate negative pressure. A cleft lip with cleft palate impairs the formation of a seal. The floor of the nasal cavity is the roof of the oral cavity. If there is a connection between them, then milk can get into the nasal cavity and be inhaled into baby's lungs (aspiration). Cleft palate can cause aspiration.

A cleft lip on its own (without cleft palate) is usually benign in the sense that the neonate will not have difficulty feeding. Cleft lip is disfiguring, however, and the emotional anguish it can cause carries with it high morbidity. Surgical correction is simple and eliminates stigma.

Salivary Glands

The salivary glands moisten food (a requirement for swallowing) and initiate chemical digestion. Because they secrete digestive enzymes into the oral cavity, the salivary glands are exocrine glands. We discuss acini in detail later in this module and want to move through this discussion quickly here. **Acinar cells** are the **columnar cells** of an exocrine acinus, a cluster of cells that secretes enzymes and mucus into a common lumen. Acinar cells secrete the stuff of saliva—enzymes, isotonic fluid with sodium and chloride, and mucus. They secrete those things into a common lumen that becomes a ductule. The cells that line the ductules (**cuboid ductal cells)** reabsorb that sodium and chloride while **secreting bicarbonate**. The ductules of the acini come together to form the ducts through which the fluid and digestive enzymes of saliva are released into the oral cavity.

The enzymes secreted by the acini chemically digest carbohydrates (**amylase**) and lipids (**lipase**). The fluid can be mucinous (thick) or serous (thin). Salivary glands are classified as mucinous (secretion is mostly mucin), serous (secretion is mostly serum), or mixed. Salivary secretion has **no hormonal control** and is almost entirely under **parasympathetic control** (ACh stimulates M_3 receptors, inducing secretion through the G_q-IP_3 pathway). The Vagus nerve carries the signal.

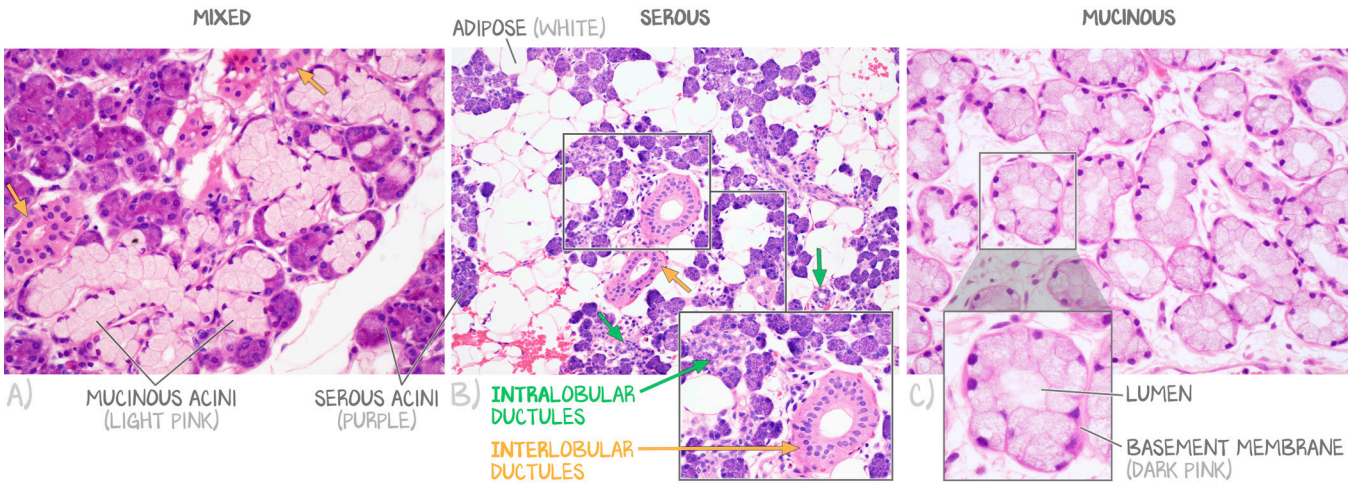

Figure 1.7: Salivary Glands
(a) High-powered magnification of the submandibular salivary gland, demonstrating both serous acini (purple) and mucinous acini (pink). Ducts can be seen at the periphery, darker pink cells surrounding a common lumen. (b) Moderate-powered magnification of the parotid gland demonstrating substantial adipose (white space) between serous glands (purple). Intralobular ducts drain the acini, and interlobular ducts drain several intralobular ducts. The cross-section of the interlobular ducts is best shown in the center of this image. (c) High-powered magnification of the sublingual gland showing an abundance of mucinous acini. The dark pink around each acinus is the basement membrane, the nuclei of the acinar cells are oriented against the basement membrane, and the apical domain of all the acinar cells of an acinus secrete into a common central lumen.

There are six salivary glands in total, three on each side of the face: parotid, sublingual, and submandibular. The **parotid** gland is serous and secretes about 25% of the salivary volume. It receives parasympathetic innervation from cranial nerve IX (CN 9, the palatoglossal nerve). The **sublingual** gland is mucinous and secretes 5% of the saliva. The **submandibular** gland is also serous and secretes the majority of the saliva, about 70%. The under-the-tongue glands are innervated by a branch of the zygomatic division of cranial nerve VII (CN 7, the facial nerve, as the **lingual nerve**). Taste sensation sensory fibers run through cranial nerve VII. The parasympathetic fibers that innervate the glands run through cranial nerve VII and synapse on ganglia near each gland. Be careful: the **facial nerve** runs **through** the parotid gland but does not innervate it. The facial nerve does innervate the sublingual and submandibular glands.

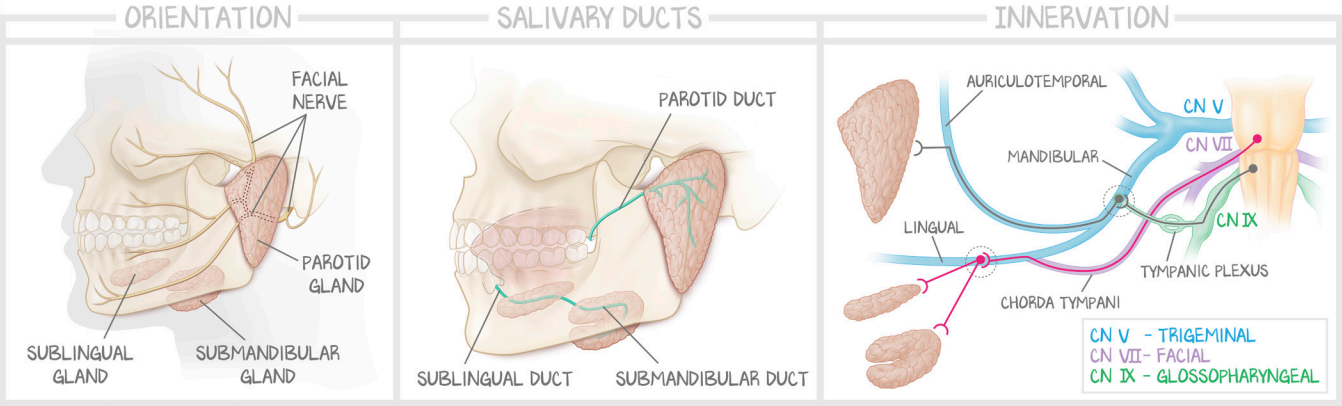

Figure 1.8: Salivary Glands
The facial nerve runs through the parotid gland but innervates the sublingual and submandibular glands. The parotid duct runs to the back of the oropharynx. The submandibular and sublingual ducts run to the base of the tongue. The crisscrossing of nerves can be confusing—branches of CN 5 conduct axons from CN 7 and CN 9. CN 7 innervates the sublingual and submandibular glands by crossing through the chorda tympani into the lingual branch of CN 5. CN 9 innervates the parotid by crossing the tympanic plexus into the mandibular branch of CN 5 then continuing through the auriculotemporal branch to the parotid.

Sialolithiasis. Sialo- (salivary gland) -lithiasis (stone) is the general term for any stone in any duct of any salivary gland. It is most common in the submandibular glands. The ducts concentrate the secretions. If they concentrate them too much, a stone can form, obstructing the duct. An obstruction causes **pain** and **swelling**, especially when the ducts are carrying saliva. This means the **pain worsens with eating**. Use NSAIDs to reduce pain and swelling, warm compresses and gland massage to push the stone along, and sour candies to promote salivary flow. Ducts get bigger the farther they are from the gland, so getting the stone to move at all generally solves the problem.

Sialadenitis. Sial- (salivary gland) -adenitis (inflammation) is the general term for any inflamed salivary gland. **Parotitis** (swelling of the parotid gland) is the most common. A single painful, swollen parotid gland is likely due to a stone or generic viral illness. Bilateral painful, swollen glands are likely due to **mumps** in an unvaccinated child. Painless swelling of the parotid gland is likely to be cancer. Because the facial nerve passes through the parotid gland, swelling (cancer) or inflammation (parotitis) can lead to paralysis or facial palsies.

Anatomy and Embryology of Tongue

The tongue enables us to speak and taste food and is responsible for sending a chewed and saliva-moistened food bolus into the oropharynx.

The development of the tongue represents a location where all three layers of the embryo are continuous with one another. It's easiest to understand if you consider the tongue from the perspective of the muscles, knowing that the tongue has taste buds, rugae, etc. The **intrinsic muscles** of the tongue (the tongue itself) serve to change the shape of the tongue, but not its location. The **anterior two-thirds** of the intrinsic muscles come from **ectoderm**. The **posterior one-third** of the intrinsic muscles is made from **endoderm**. The **extrinsic muscles** of the tongue (those that attach to the tongue and originate from a bone) are the muscles that change the placement of the tongue, but do not alter its shape. The extrinsic muscles, like all skeletal muscle, are derived from **mesoderm**.

Tongue innervation is complex and involves branches from several cranial nerves. The anterior two-thirds have taste from CN 7 (CN VII, facial nerve, as the chorda tympani to the lingual nerve) and sensory from CN 5 (CN V, trigeminal, V3 division, as the lingual nerve). A branch of CN 5 and a branch of CN 7 run together as the lingual nerve. The posterior one-third of the tongue is innervated for taste and sensation by CN 9 (CN IX, glossopharyngeal). The epiglottis and palate have taste and sensation from CN 10 (CN X, vagus). Motor innervation of all intrinsic muscles of the tongue comes from CN 12 (CN XII, hypoglossal nerve).

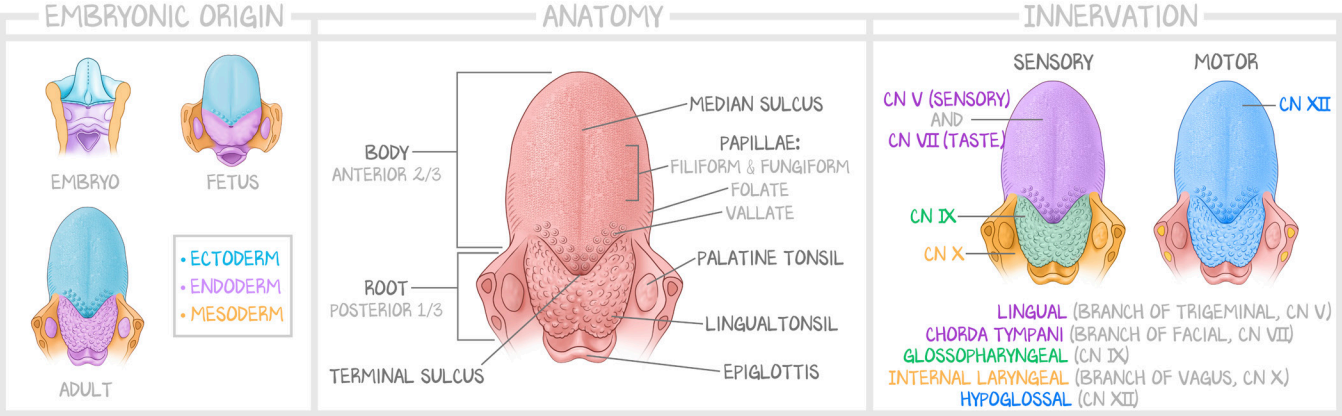

Figure 1.9: Tongue Anatomy and Innervation
Using what you saw in Fig. 1.7, you can follow the development of the tongue from in utero to adult. We removed the color-coding in the anatomy panel. The anterior two-thirds of the tongue is called the body and is derived from ectoderm. The posterior one-third of the tongue is called the root and is derived from endoderm. Compare Fig. 1.10 to this illustration to see the connections. Finally, the innervation of the tongue is a little tricky. The body is innervated by CN 5 (sensory), CN 7 (taste), and CN 12 (motor). The root is innervated by CN 9 (taste) and a smidgen by CN 10 (taste and motor).

All extrinsic muscles except the palatoglossus receive motor innervation from CN 12 (CN XII, hypoglossal). That means there is one motor supply (CN 12) to every muscle involving the tongue—intrinsic or extrinsic—except the palatoglossus muscles, which are innervated by CN 10. The palatoglossus muscles are beneath the mucosa of the palatine arches. More on this later in this lesson.

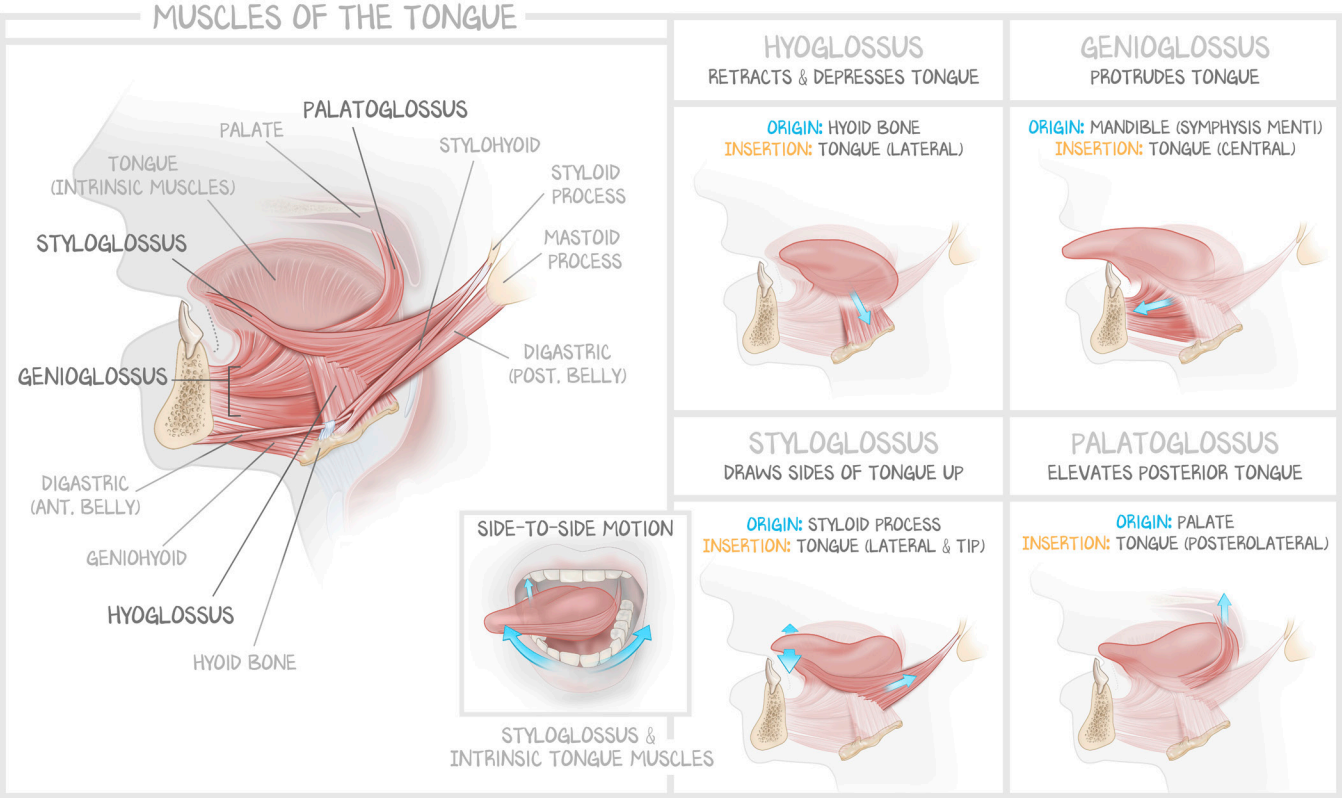

Figure 1.10: Muscles of the Tongue
The details of this illustration are not necessary to commit to memory. It attempts only to show you that their names are merely a combination of the tongue (glossus) and the bony processes they originate from.

Swallowing

Oral phase, pre-swallow phase. This is the phase where the teeth grind up the food (mechanical digestion), the salivary enzymes break down the food (chemical digestion), and the salivary juices enable the formation of a food bolus. The **facial nerve** (CN 7) innervates the skeletal muscle of the face and mouth. A person chews their food, the grinding and mastication all under somatic control, while the salivary glands produce saliva. Salivation is not under somatic control. The facial nerve runs through the parotid gland but does not innervate it. Once you want to swallow and the food has been made into a food bolus, you start the swallow.

Oral phase, swallow. You decide the bolus is ready. The oral phase is under somatic, **voluntary control**. When you decide you want to swallow (do it, right now, swallow some saliva or a nearby drink), the intrinsic muscles of the tongue **shape** the food bolus by curling up. The extrinsic muscles yank the base of the tongue backward and lift the posterior of the tongue even further. You smash the tip of your tongue and the food bolus against the palate. And, with nowhere else to go, the **food bolus gets propelled backward** into the pharynx.

It hits the oropharynx. Sensory fibers lie in a ring around the pharyngeal opening, with the greatest sensitivity at the tonsillar pillar. They feel the food bolus and transmit the signal up the **glossopharyngeal** nerve (CN 9). To the nucleus tractus solitarius the signal goes. From the **swallowing center** in the **medulla** come the motor impulses for the successive phases of swallowing. Those signals start by opening the esophagus and closing everything else. The oral phase is over, and the pharyngeal phase has begun.

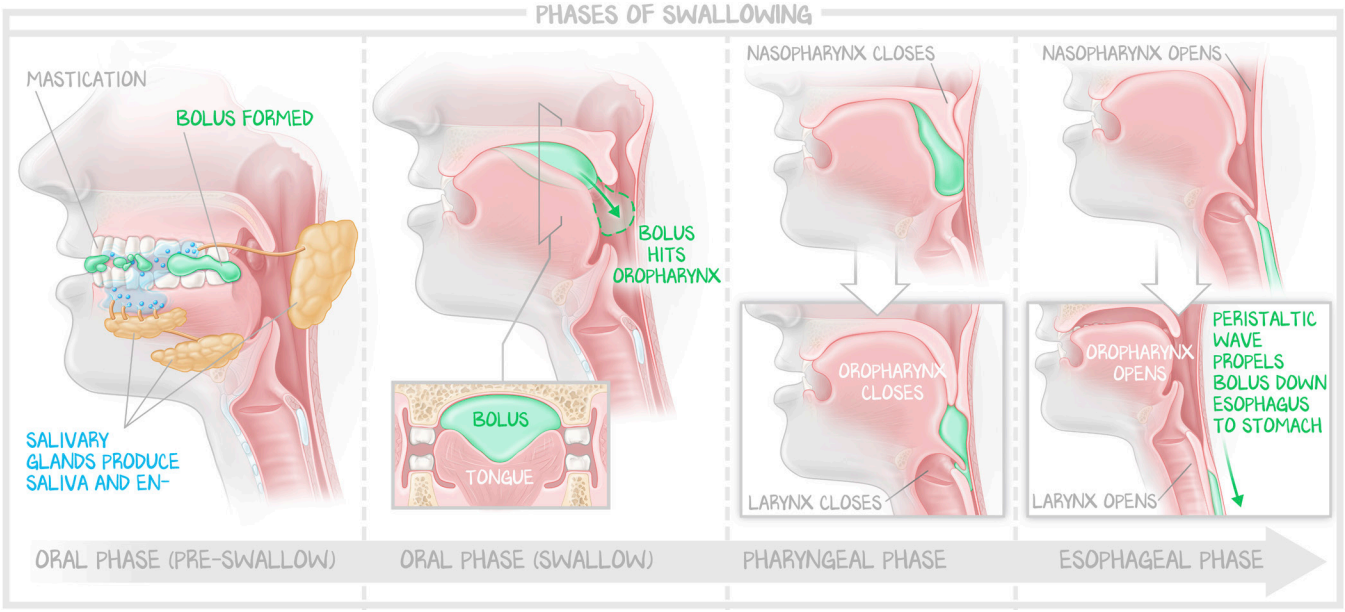

Figure 1.11: Phases of Swallowing
The oral phase involves mastication, salivation, and the decision to swallow. The pharyngeal phase begins as the food bolus is passed through the palatine arches, and the food bolus hits the oropharynx. At that point, a coordinated reflex closes the epiglottis and opens the upper esophageal sphincter. The esophageal phase is continuous with the pharyngeal phase, and a coordinated peristaltic wave propels food to the stomach.

Pharyngeal phase. The food bolus is in the oropharynx. You want food to go to the esophagus and stomach. You do not want food going down the trachea or up into the nasopharynx. This phase is **involuntary**. The oropharynx's "in" hole is closed off by your tongue—the same muscles that initiated the backward movement now prevent the food from moving forward. The **nasopharynx** closes by upward retraction of the **soft palate**. Snarfing milk when you laugh while drinking is a failure of this reflex. The palatopharyngeal arches on each side of the pharynx pull medially, effectively creating a slit for the food to pass through. Food that is not well chewed will not fit well. This is why it hurts when you try to swallow that steak you didn't chew well. The larynx closes in a redundant mechanism (which is smart because food in your lungs is bad)—the **closure of the true cords** by the arytenoid muscles (airway muscles close off the airway) and the **downward contraction of the epiglottis** (oral muscles close off the airway). At the same time, as these muscles close off the airway, the **hyoid muscle** elevates the larynx higher into the pocket made by the epiglottis. This lift and simultaneous relaxation of the upper esophageal sphincter widen the opening to the esophagus.

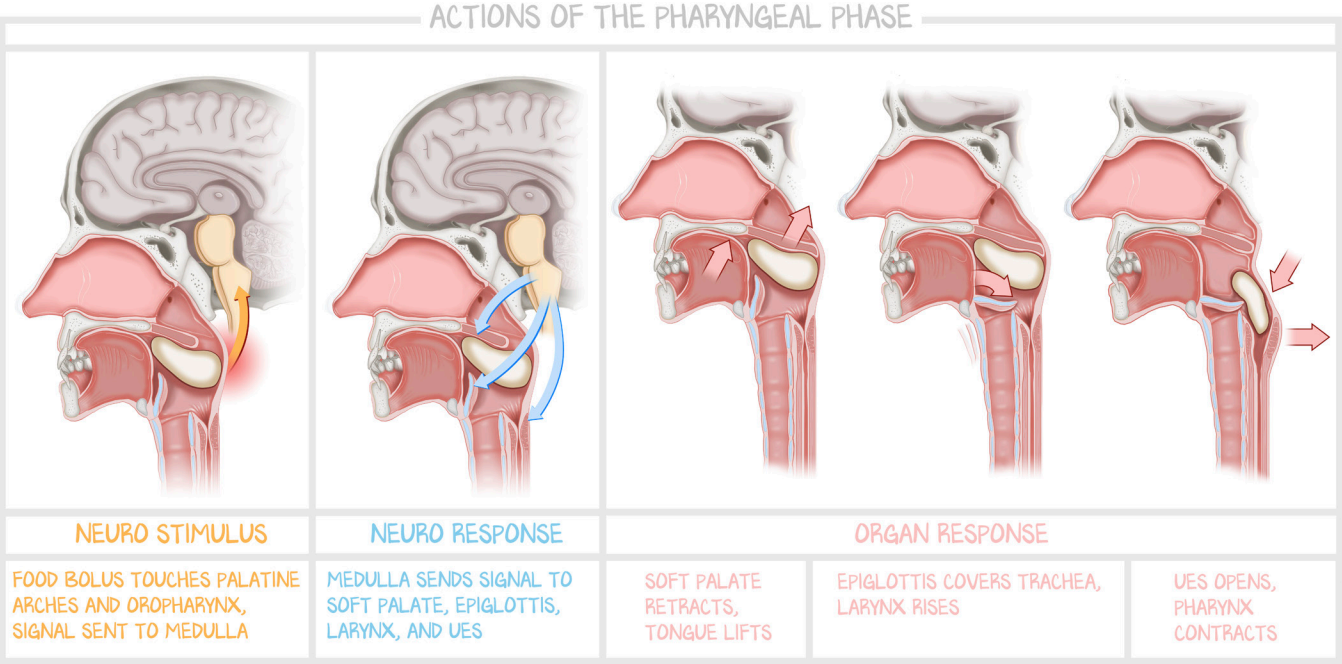

Figure 1.12: Actions of the Pharyngeal Phase
The food bolus is propelled against the oropharynx. Receptors on the pharyngeal arches and oropharynx send the afferent (sensory) information to the medulla, activating it. In response, the medulla sends a signal through the efferent (motor) fibers, initiating the involuntary swallowing reflex. This is coordinated and nearly simultaneous, although we have separated the organ response into the soft palate, epiglottis, and the upper esophageal sphincter (UES). The soft palate lifts to block the nasopharynx, the epiglottis closes to protect the airway, and the UES opens.

Esophageal phase. We talk about peristalsis and the opening of the LES in the next lesson on the esophagus.

Oropharyngeal Dysphagia

Problems must lie with the non-esophageal phases of swallowing—either the oral (voluntary) or pharyngeal (involuntary) phases. Technically suckling (discussed under cleft lip and palate) is an oropharyngeal dysphagia. However, we want you to think about oropharyngeal dysphagia as a **neural problem** of the oropharynx. Speech therapy (ST in hospital admissions) assesses cognition, speech, and swallowing. ST consultation is a mandatory part of any stroke evaluation. To coordinate so many muscles requires so many nerves (cranial nerves 5, 6, 7, 9, 10, 11, and 12), and those nuclei are so spread out that any damage to any of them may lead to problems with swallowing. Patients with oropharyngeal dysphagia will have trouble **initiating swallowing**, presenting with **coughing** or **choking** at the very beginning of swallowing. This is opposed to esophageal dysphagia, which will occur after initial swallowing has been achieved. What you care about in oropharyngeal dysphagia is **aspiration**. All those mechanisms are designed to keep food out of the trachea, but in patients with impaired neurology—stroke, Parkinson's, myasthenia gravis, multiple sclerosis—any of those mechanisms may not work. And "not work" may mean the aspiration of large foods, soft foods, or even liquids.

A **videofluoroscopy** is diagnostic. A swallow study is performed with varying grades of substances—water, thickened liquids, whole food—and the phases of mastication and subsequent movement of the bolus towards the oropharynx and where it ultimately ends up (in the esophagus or trachea) can all be seen on radiography.

Citation

Figures 1.7a, 1.7b, 1.7c: Courtesy of WebPathology.